Sarcocystis cernae

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Sarcocystis cernae: A parasite increasing the risk of predation of its intermediate host, *Microtus arvalis*

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Summary. 1) The transmission dynamics of the protozoan parasite Sarcocystis carnae (Černá and Loučková 1976) (Apicomplexa, Eimeroidea, Sarcocystidae) in natural populations were studied in the Lauwersmeerpolder in the northern Netherlands. This parasite needs two hosts to complete its life cycle; the common vole (Microtus arvalis) as its intermediate host and the kestrel (*Falco tinnunculus*) which preys on the vole, as its final host. 2) Seasonal variation in prevalence of infection in snap-trapped common voles was determined in two years, 1984 and 1985. It was found to be lowest in November (6% of the voles infected) and it increased gradually to a peak in May (33%). 3) Data collected in three successive kestrel breeding seasons (1983-'85) revealed that voles in the kestrel summer diet are infected twice as frequently as those in snap-trap samples, 21% and 9% respectively. This difference ($P<0.05$, X^2-test) suggests that the parasite influences its intermediate host behaviour in such a way that it enhances the probability of parasite transmission to the final host.

Key words: Sarcocystis – Microtus – Kestrel – Predation risk – Parasite

Many parasites with alternating hosts depend on predation of the intermediate host by a final host for the completion of their life cycle. Such parasites would benefit from mechanisms promoting the specific risk of the intermediate host, as suggested by Holmes and Bethel (1972). Effects of parasites on host behaviour have repeatedly been reported (Carney 1969; Wickler 1968; Bethel and Holmes 1973, 1977; Camp and Huizinge 1979; Brown and Tompson 1986). In these studies – except Bethel and Holmes 1977 – no evidence was presented that the behavioural changes observed actually increased the vulnerability of parasitised prey. Moore (1983, 1984a, b) has shown in an extensive study increased vulnerability to bird predation of isopod prey parasitised with acanthocephalan parasites.

We have analysed vulnerability of intermediate hosts for the protozoan parasite Sarcocystis cernae. This parasite spends its life cycle in two hosts, the intermediate host, the common vole, (Microtus arvalis), and the final host, the kestrel, (Falco tinnunculus) (Černá and Loučková 1976; Tadros and Laarman 1976). The common vole is the main prey in the diet of kestrels in the Netherlands (Cavé 1968).

S. cernae belongs to the tissue invading Coccidia (Family Sarcocystidae) of which several life cycli have been elucidated in laboratory experiments (Levine and Tadros 1980), summarized for some voles and mice in Table 1. All these parasites are adapted to a specific predator-prey relationship.

The transmission cycle comprises four stages (Fig. 1). A kestrel infected with S. cernae sheds sporocysts from its small intestine with the faeces. When orally ingested by a common vole, the parasite develops in the liver where it undergoes asexual multiplication followed by invasion of the musculature by free merozoites in the blood stream. In the muscles the parasite reproduces and forms large sarcocysts containing numerous cystozoites (8 × 2 micrometer). The cystozoites are liberated from the sarcocysts when an infected vole is eaten by a kestrel upon which they immediately complete sexual development in the subepithelial layer of the small intestine of the kestrel, leading to the formation of sporocysts (Černá and Loučková 1976). An infected kestrel is able to shed infectious faeces in the environment about 14 days post infection (W.D. Tadros, unpublished work). The sporocysts kestrels excrete, when infected, are known to be very resistant stages in the parasites life cycle. They are small, oval structures (13 × 10 micrometer), notably resistent to adverse environmental conditions. In an aqueous laboratory environment they can survive more than a year (Long 1982). Although the life cycle of S. cernae has been elucidated in general, little is known about the dynamics of this parasite in the field.

This study reports data collected on the parasite transmission from intermediate host to final host. The research was carried out in the Lauwersmeerpolder (53°20′N, 60° 12′E) in the Netherlands in 1983 till 1985 and was made possible by a simultaneous research project on predator-

Table 1. Four sarcocystis parasites with developmental stages in mice, voles and their predators (From Levine and Tadros 1980)

Parasite	Intermediate host	Final host
S. cernae	Microtus arvalis	Falco tinnunculus
S. sebeki	Apodemus sylvaticus	Strix aluco
S. dispersa	Mus musculus	Tyto alba
S. putorii	Microtus arvalis Microtus agrestis	Mustela nivalis

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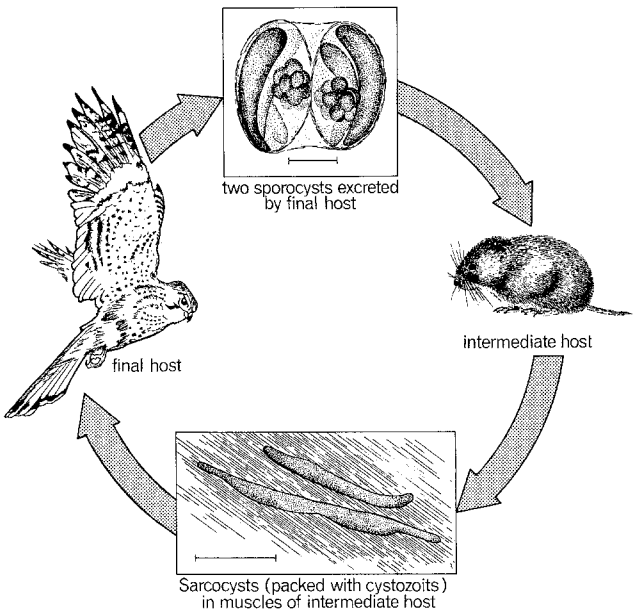

Fig. 1. The life-cycle of Sarcocystis cernae

prey relationships in the kestrel and common vole going on in this area (see e.g. Rijnsdorp et al. 1981 and Masman et al. 1986a). The analysis of parasitic infection in common voles allowed me a) to evaluate the seasonal variation in S. cernae prevalence in relation to kestrel and vole densities and b) to compare the prevalence of S. cernae among kestrel-caught voles and snap-trapped voles as indicative of relative predation risks.

Methods

Field observations on kestrels and snap-trappings of common voles were carried out in the Lauwersmeerpolder (9000 ha). About 40 nestboxes are available to kestrels throughout this area.

The analysis is based primarily on three sets of data:

1) Trapped voles, standard census

To provide background information on vole densities in the study area, a standard trap census has been carried out for over five years in two-monthly intervals. 500 Snap-traps baited with carrot were distributed over 10 standard plots in the area. Traps were checked for three consecutive days. Voles trapped were sexed and weighed. Vole weight is provisionally considered as a parameter for vole age. The census was started in March 1981 as part of the kestrel project. A total of 403 snap-trapped voles were analysed for S. cernae of which 336 weighed 14 grams or more.

2) Voles caught by kestrels

In the kestrel project, a number of experiments concerning parental investment were performed, in which brood satiation was manipulated by removal of prey delivered to the nest (Masman et al. 1986a). These experiments were usually done in May (the last two weeks), June or July, depending on the age of the young kestrels. Nestboxes of some selected kestrel pairs were mounted against a hide and opened on the back. An observer behind the nest recorded all prey items brought to the young kestrels. Prey items were weighed and depending on the experiment either removed from the nest or replaced by labmice of the same weight. In addition to these prey, 46 voles were collected in May till July 1984 during weekly inspections of kestrel nestboxes. Altogether 422 collected kestrel prey were analysed of which 346 voles weighed 14 grams or more.

3) Trapped voles, around kestrel nestboxes

Voles were snap-trapped to obtain data on the prevalence of infection of S. cernae in voles living in the hunting areas of kestrels of which the prey had been sampled from the nestbox. Between 350–550 snap-traps were placed in the months May till July at appropriate sites where we observed kestrels catching voles. Traps were placed at entrances of burrows and in runways with signs of vole activity present (such as fresh faeces and little pieces of grass left after foraging). Traps were checked daily for 7–14 days in order to trap a large number of voles. 222 Trapped voles were analysed for S. cernae of which 166 voles weighed 14 grams or more.

All voles collected were either stored at $-20°$ C in plastic bags until later analysis or analysed directly. A number of voles in which Sarcocystis had been detected in fresh analysis were stored in the freezer and later reanalyzed. No deleterious effects on parasite visibility due to freezing were found.

Voles were analysed for infection with S. cernae by careful examining fresh squash samples of six different muscles under a light microscope, using 10×10 magnification. The following muscles were analysed: three leg muscles: musculus biceps, m. triceps and m. quadriceps; two rump muscles: m. pectoralis and m. spinotrapezius and both chewing muscles: m. masseter. Sarcocysts of S. cernae could easily be recognized among the muscle fibres. They consist of elongate structures divided into numerous compartments filled with cystozoites (Černá and Loučková 1976). Differences between infected and non-fected voles (for instance in colour or physical appearance) are not obvious to human eyes.

Sarcocyst sizes vary with the stage of infection. They range from 50 to 8000 micrometer in length. In old infections (sarcocysts sizes about 500 micrometer long and 250 micrometer wide), sarcocysts could be seen macroscopically in the swollen muscular tissue. The red muscles appeared white-striped due to the cysts lying parallel to the muscle fibers. When making fresh squash slides, sarcocysts may break but parts of cysts scattered in the muscles still can be recognized. A vole was considered infected when at least in one muscle sample a sarcocyst or parts were found. Most voles examined for infection were complete animals. A total of 1047 voles was analysed of which 848 weighed 14 grams or more and 85 of the 1047 were found infected with S. cernae. Some voles were incomplete (110 of the $1047 = 10.5\%$) which means that they missed head, rump or various legs so not all six muscles could be analysed.

Besides S. cernae sometimes *Sarcocystis putorii* was detected. The final host for this parasite it the weasel, *Mustela nivalis* (Tadros and Laarman 1982). S. cernae and S. putorii can be distinguished from each other by morphologically different sarcocyst walls. S. putorii has short blunt cyst wall projections, which S. cernae lacks. Mixed infections of S. cernae and S. putorii were found in 13 of 848 adult voles

Table 2. Fraction of muscles infected of voles in which at least one sarcocyst was found. Voles weighed 14 grams or more and all six muscles had been analysed

	Infected voles, caught in snaptraps, $n=35$	Infected voles, caught by kestrels, $n=25$	All infected $n=60$
Musculus biceps	66%	76%	92%
Musculus triceps	80%	88%	
Musculus quadriceps	63%	60%	
Musculus pectoralis	60%	52%	67%
Musculus spinotrapezius	37%	16%	
Musculus masseter	40%	24%	30%

(=1.5%). In 6 of the 848 voles (=0.7%) infections with only S. putorii were found. The frequency of mixed infections was significantly higher than would be expected from independent occurrence ($X^2=67{,}0$, $P<0{,}001$, df=1).

Because it was important to estimate the age of infection, a growth curve of S. cernae in voles was constructed. This was done in 1982 by infecting 23 laboratory-reared common voles with S. cernae sporocysts collected form natural kestrel faeces. Voles were killed after successive days post infection and sarcocyst lengths in their musculus pectoralis were measured after isolation from the muscles by enzymatic procedures (C.A.M. Koenis and J.J. Laarman, unpublished work).

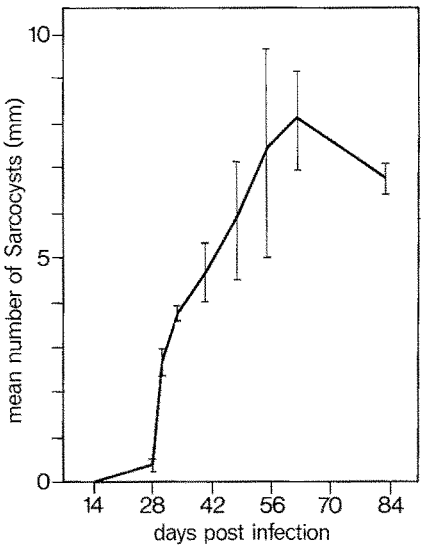

Fig. 2. Growth curve of S. cernae in experimentally infected common voles ($n=23$). Unpublished data C.A.M. Koenis University of Amsterdam (1982). I mean S.D.

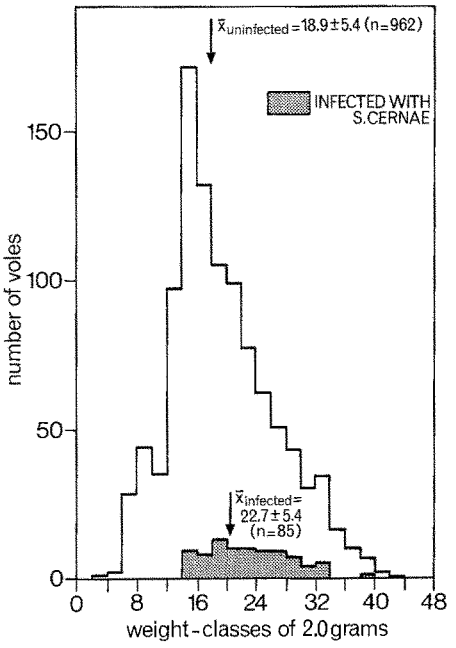

Fig. 3. Weight distribution of Microtus arvalis analysed ($n=1047$). Indicated are infected voles ($n=85$) and uninfected voles ($n=962$) and mean weights

Results

Distribution of S. cernae in the vole muscles, the relation of infection with vole age and sex

The frequency of occurrence of S. cernae in the six muscles analysed is shown in Table 2. In 92% of the infected voles S. cernae was found in at least one of the three locomotory muscles (musculus biceps, m. triceps and m. quadriceps). In 67% of the infected voles S. cernae was found in one or both rump muscles (M. pectoralis and m. spinotrapezius). In only 30% of the infected voles S. cernae had invaded the chewing muscles (m. masseter). This can be interpreted as selection of the locomotory muscles by the parasite. The development of S. cernae within vole muscles is shown in Fig. 2. About a month after infection S. cernae can be found microscopically in the musculature. Of all the 1047 voles analysed no voles weighing less than 14 grams were found to be infected with S. cernae. This can be explained by the development of young voles and the parasite. After birth it takes about three weeks before young voles weight circa 9 grams (Reichstein 1964). After weaning at this age, the young voles leave the nest of birth and come above ground to start foraging for themselves. From that moment onwards there is a risk that they come in contact with kestrel faeces containing S. cernae sporocysts. Taking into account the developmental growth of S. cernae the chance that voles weighing less than 14 grams harbour S. cernae must be very small. Therefore voles weighing less than 14 grams were omitted from the final calculations. The prevalence of infection in various weight-classes (roughly indicating age) of voles is shown in Fig. 3. The weight of the lightest infected vole was 14.5 grams and the heaviest infected vole weighed 38.0 g. Mean weight of infected voles is significantly higher than that of non infected voles, ($t=6.22$, $P<0.01$, df=1043). In Table 3 the fraction of males and females infected are shown. Of the infected voles caught by kestrels, infected males were significantly more caught than infected females ($X^2=5.79$, $P<0.025$, df=1). No such difference was found in voles caught in snap-traps.

Seasonal fluctuation of prevalence of S. cernae in voles in the field

The infection rate of S. cernae in the field throughout two years was determined by analysing samples of voles trapped

Table 3. Prevalence of S. cernae in female and male voles, weighing 14 grams or more

Voles caught in snap-traps, traps placed in hunting areas of kestrels, May till July

Year	Total analysed	♀♀	♂♂	Sexe-ratio	Total infected	% ♀♀ infected	% ♂♂ infected
1983	17	6	11	65%	1	17%	–
1984	80	31	49	61%	13	13%	18%
1985	107	52	55	51%	5	6%	4%
1983–1985	204	89	115	56%	19	9%	10%

$X^2 = 0.01$, $P > 0.05$, df = 1

Voles caught by kestrels, May till July

Year	Total analysed	♀♀	♂♂	Sexe-ratio	Total infected	% ♀♀ infected	% ♂♂ infected
1983	26	12	14	54%	12	42%	50%
1984	76	32	44	58%	18	9%	34%
1985	87	49	38	44%	9	8%	13%
1983–1985	189	93	96	51%	39	13%	28%

$X^2 = 5.79$, $P < 0.025$, df = 1

Voles caught in snap-traps and by kestrels; voles collected throughout the year

Year	Total analysed	♀♀	♂♂	Sexe-ratio	Total infected	% ♀♀ infected	% ♂♂ infected
1983	170	85	85	50%	20	12%	12%
1984	265	118	147	56%	45	12%	21%
1985	388	183	205	53%	20	5%	5%
1983–1985	823	386	437	53%	85	9%	12%

$X^2 = 2.14$, $P > 0.05$, df = 1

in the standard census between July 1983 and July 1985. The lowest prevalence of infection occurred in November (5% in 1984 and 8% in 1985, see Fig. 4). Gradually the infection rose to a peak in May (25% in 1984 and 67% in 1985). This last percentage however is based on the very low sample size of 3 voles. To understand how infection with S. cernae can fluctuate throughout the year we must pay attention to the seasonal change in vole and kestrel numbers.

Changes in vole numbers

Vole numbers fluctuate seasonally from a minimum in May to a maximum in September (Fig. 5). The pattern was the same each year but densities varied between years. 1983 Was a year with high vole numbers while in 1984 density was low. These fluctuating numbers contain vole cohorts born at different times of the year (Fig. 6). This figure is a result of one year live-trapping in the Lauwersmeerpolder in 1979/'80 and is thought to give a good representation

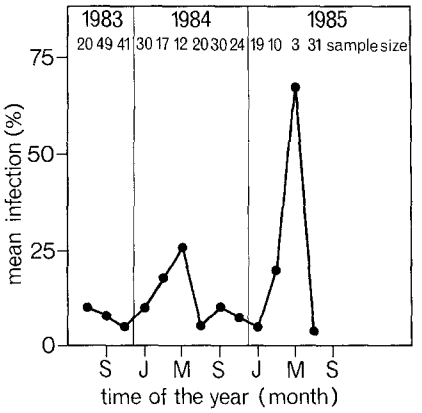

Fig. 4. Prevalence of infection with S. cernae in voles. Analysis of voles which were snap-trapped between July 1983 and July 1985 in the standard-trap census

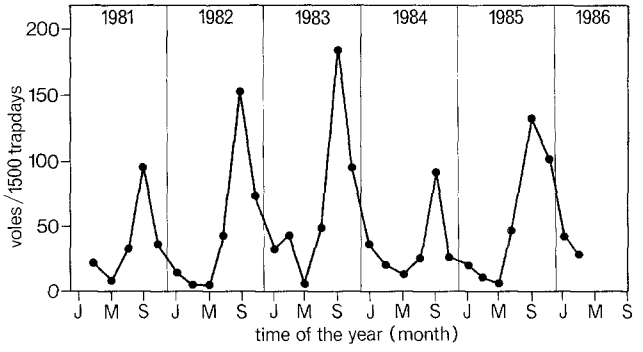

Fig. 5. Yearly fluctuations in vole numbers. Voles snap-trapped in standard trap census bimonthly from 1981 onwards

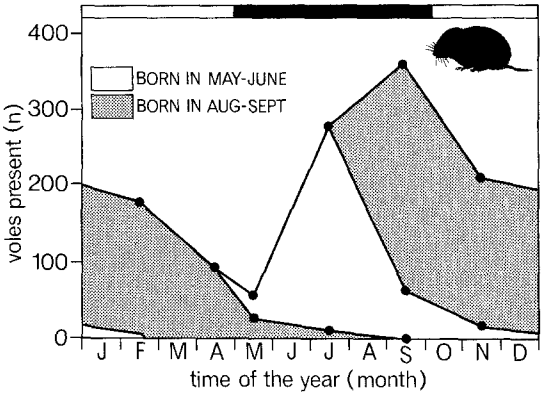

Fig. 6. Seasonal fluctuations and composition of vole numbers. Data from one year live-trapping on a 05 ha. plot. For details of methods see Hoogenboom et al. 1984. Black bar: breeding season

of the composition of vole populations (see for details Hoogenboom et al. 1984). Indicated are two different cohorts. A cohort is here defined on the basis of the initial trapping date. Voles were marked individually, released and recaptured. When voles are live-trapped no technique exists to reliably estimate date of birth of those trapped as adults only. We can only guess roughly when juvenile voles were born. If marked voles are not recaptured after some months they may be dead or have emigrated. Our longest trap record of the same vole was one and a half year. But as shown in Fig. 6 most voles are not recaptured six months

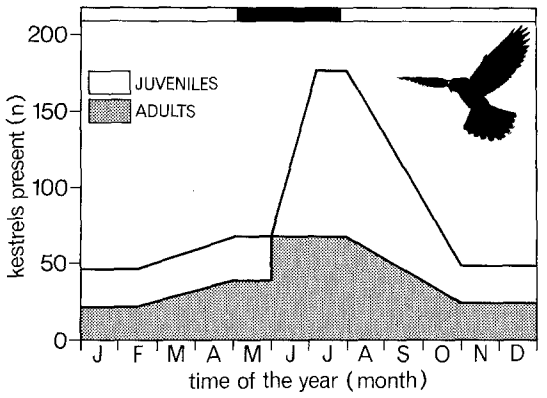

Fig. 7. Seasonal fluctuations in kestrel numbers. (After Masman et al. 1986a). Black bar: breeding season

after first capture. Voles which survied the previous winter start to reproduce in April. Their offspring reproduces in July–August. This second cohort, born in July–August, usually does not start to reproduce until the following spring.

Changes in kestrel numbers

Kestrel numbers also fluctuate throughout the year. Figure 7 shows that a limited number of adults and juveniles stay in the polder during winter. They start breeding between May and June. The eggs hatch about one month after egg laying. The number of kestrels present in the Lauwersmeerpolder increases in spring first by immigration and then due to fledging in the locally born birds in June–July. Juvenile birds never breed in the year of their birth but frequently breed the following year. Kestrels mainly hunt for voles (Cavé 1968). Their hunting consists primarily of flight-hunting in the air. They make strikes towards the ground to catch voles. Voles are not continuously active above ground. They exhibit a circa two-hour rhythmicity during the day (Daan and Slopsema 1978; Raptor Group 1982 and Hoogenboom et al. 1984). Kestrels have been shown to synchronise their hunting activity with vole activity (Rijnsdorp et al. 1981).

Are Sarcocystis infected voles more prone to kestrel predation?

In order to analyse if infected voles have an increased vulnerability for predation by kestrels, a comparison was made between the percentage infected among voles caught by kestrels and among those caught in snap-traps. This is only possible to do in the breeding season (May till July/August) when the parent kestrel(s) bring voles to their young in the nestbox. Only during these months we were able to obtain adequate numbers of voles by manipulating nestbox deliveries. In the early breeding season some voles could also be collected from nestboxes during weekly inspections. In other times of the year it was only possible to obtain a kestrel-caught prey if we actually saw a kestrel caching a vole instead of eating it. Then it was possible to search for the voles cached in the vegetation. In November 1983 and in January 1984 four voles caught by kestrels could be analysed in that way.

Table 4 shows data of three successive breeding seasons in which voles could be sampled and which were analysed

Table 4. Prevalence of S. cernae in voles caught in traps and by kestrels, weighing 14 grams or more. Traps placed in hunting areas of kestrels, May–July

Year	Voles caught in snap-traps	Infected *n*	%	Voles caught by kestrels	Infected *n*	%
1983*	17	1	5.9%	26	12	46.2%
1984	80	13	16.3%	76	18	23.7%
1985	107	5	4.7%	87	9	10.3%
1983–1985**	204	19	9.3%	189	39	20.6%

* $X^2=6.1$, $P<0.025$, df=1
** $X^2=9.1$, $P<0.005$, df=1

for S. cernae. The three years combined showed a significant difference between the prevalence in kestrel-caught and trap-caught voles ($X^2=9.1$, $P<0.005$, df=1). More infected voles were caught by kestrels than would be expected from the prevalence of infection in the hunting areas of the kestrels. For each year separately the same trend appeared although the difference was significant only in 1983. Thus Sarcocystis infected voles were overrepresented by a factor two in the kestrel-caught samples as compared to the trapped samples. Either the traps selected for non-infected voles or the kestrels selected for infected voles.

Discussion

The dynamics of transmission of S. cernae in the field

The prevalence of S. cernae in voles fluctuated seasonally (Fig. 4). By what factors could this be caused? To answer this question we must look at population changes in the predator, the prey and the development of the parasite itself.

At the end of the summer, beginning of autumn, the largest number of (mostly young) voles is present, the second cohort of that year. 6% Of these voles were found infected with S. cernae. After the winter, in May, the percentage voles infected was much higher: 33%. This can be explained by the presence of the largest proportion of old animals in the population at that time which have the greatest chance of carrying a fully developed infection. The parasite needs about a month (Fig. 2) to become microscopically visible in the muscles. Thus the chance that a vole harbours a parasite becomes larger in the autumn, winter and spring months.

The number of kestrels is the highest in the summer months. Then many pairs have young to raise by catching voles for them. Parent kestrels select the prey which they bring to their young. The light voles (mean=11.5±4.1 grams) they often eat themselves, while voles brought to the nest are somewhat heavier (mean=16.1±7.1 grams) (Masman et al. 1986b). These heavier voles are more infected than the young voles, so the chance for young kestrels to eat infected prey may be slightly larger than for the parent kestrels. Not all birds will become infected, depending on their protective immune system and general condition. But young kestrels have not yet developed a specific immunity to sarcocystis, while older birds may have

Table 5. Intermediate host behaviour affected by parasitic infection

Parasite	Intermediate host	Final host	Changed intermediate host behaviour
Polymorphus paradoxus	Gammarus lacustris	Anas plathyrhynchos	Altered photic and/or evasive behaviour
Polymorphus marilus	Gammarus lacustris	Aythya affinis	Altered photic and/or evasive behaviour
Diplostomum spathaceum	Leuciscus leuciscus	Larus ridibundus	Changed orientation and feeding behaviour
Leycochloridium sp.	A snail species	A bird species	Pulsating tentacles
Dicrocoelium dendriticum	Ants	Sheep	Changed orientation behaviour
Acanthocephallus dirus	Asellus intermedius	Leuciscus cephalus	Changed orientation behaviour; altered colour
Pomphorhynchus laevis	Gammarus pulex	Barbus barbus	Altered photic behaviour; altered colour
Plagiorhynchus cylindraceus	Armadillidium vulgare	Sturnus vulgare	Changed orientation

[a] Bethel and Holmes (1973, 1977); [b] Crowden and Broom (1980); [c] Wickler (1968); [d] Carney (1969); [e] Camp and Huizinge (1979); [f] Brown and Thompson (1986); [g] Moore (1983)

aquired this specific immunity. Little is known about the development of protective immunity in kestrels against S. cernae. On the development of protective immunity against *Frenkelia microti* – a vole brain invading protozoan parasite related to Sarcocystis – experiments are done. Common adult buzzards (final hosts) infected with bank voles containing F. microti showed protective immunity after the second and following infection attempts (Tadros and Laarman 1982).

Kestrels have specific sites in the field (poles, trees, traffic-signs), houses) where they rest, eat and defecate. On the gound under these sites concentrations of contaminated faeces may pile up. When in the nest, young kestrels typically defecate outside the nest, standing on the edge of it. There are indications that infection with S. cernae has a local distribution (C.A.M. Koenis unpublished work; this study). In 1983, a pair of kestrels which caught 46.2% infected voles (see Table 4) caught them in one small part of their large hunting area. In 1984 and 1985 no clear local concentration of infection in kestrel-caught voles was observed. This was probably caused by a much lower density of voles present those years (Fig. 5). Low vole densities forced the kestrels to extend their hunting areas and to catch voles in many different sites, located far from each other.

The effects of the parasite on the kestrel are not well known. There are indications that when the birds are in a poor nutritional state, the parasite may have fatal consequences. In 1975, when vole densities were low, many juvenile kestrels were found dead in The Netherlands containing sporocysts in their small intestines (J.J. Laarman, unpublished work).

In the field voles probably take in sporocysts while eating grass or while digging in the earth and grooming activities thereafter. Vole populations living near sites where kestrels defecate infected faeces have a bigger chance to become infected than voles living far from such sites. How many infected sites occur should depend each year on the number of infected kestrels present. Voles just after weaning (weighing 14 grams or more) start foraging grass above ground. When voles become sexually active, the home range of females and males changes. Pregnant and lactating females show reduced home ranges while sexually active male voles expand their home ranges considerably (Mackin-Rogalskie 1979). Hence, one might expect that the chance to become infected differs between females and males in the breeding season. In winter/autumn, the nonbreeding season, such chance differences will be much less because differences in home range size and behaviour between females and males gradually disappear. However, differences between percentage infected snap-trapped male and female voles in the breeding season were not found (10% and 9%, Table 3). Male and female voles were caught in the same frequency by kestrels and snap-traps (mean sexe-ratios 51% and 56%, table 3). The reason that kestrels catch more infected male voles than infected females can also not be found in higher weights of male voles. Infected kestrel caught male voles had a mean weight of 22.4 ± 5.2 gram ($n=27$) and female voles 25.5 ± 6.4 gram ($n=12$), while infected snap-trapped males weighed 24.2 ± 4.4 gram ($n=11$) and females 23.5 ± 5.2 gram ($n=8$).

Possible effects of S. cernae on intermediate and final host

The effect the parasite seems to have on its intermediate host is to increase the vulnerability of infected voles for predation by kestrels. As shown earlier in Table 4, infected voles have a twice as large chance to be caught by kestrels than would be expected from the prevalence of infection in the trapped voles. By what factors could this be caused? From the literature several examples of changed behaviour of infected intermediate hosts are known (Table 5). We can only speculate about the nature of changed vole behaviour. There are at least three hypothetical explanations:

1) When infected, a vole has to spend more time above ground than a not infected vole,

2) An infected vole is active at times of the day with enhanced predation risk,

3) An infected vole escapes more slowly when attacked by a kestrel than an uninfected vole.

S. cernae invades especially the locomotory muscles as was shown in table 2. Therefore it may be hypothesised that the movements of infected voles may be impeded. We surmise that infected voles move slower or in other ways differently in comparison with non-infected voles. Their chewing muscles are also often (but much less) infected. This might mean that they need to forage longer or more frequently than non-infected voles because chewing maybe goes slower. Voles are strict herbivores and forage every 100–150 min above ground under day hours (Hoogenboom et al. 1984).

The functional benifit to the parasite of enhanced vulnerability of its intermediate host to predation by its final host, is that it ensures the parasites completion of its life cycle. If the parasite is able to manipulate intermediate host behaviour in this way, it enhances parasite transmission.

The benefit for the predator in this situation could be that infected prey gives a higher hunting yield. Flight-hunt-

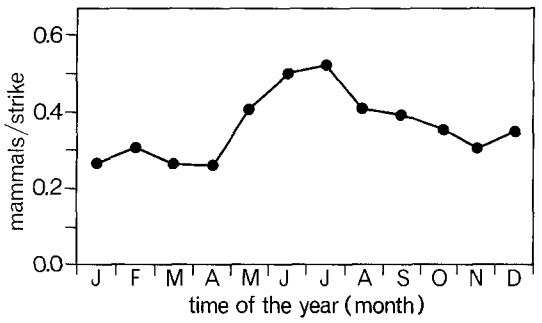

Fig. 8. Strike-success of kestrels throughout the year. (From Masman et al. 1986a)

ing by kestrels is the main hunting mode throughout the year. Strike-success varies with time of year (Fig. 8). It is not excluded that the increase in May and June is explained by the high % of infected voles: changed behaviour of infected voles may make them easier prey for hunting kestrels, although also more juveniles and pregnant female voles in spring might contribute to increased strike-success.

The intermediate host harboring S. cernae seems to suffer most from being infected with Sarcocystis cernae. Their movements are hampered by the presence of the parasite and their chance to die because a kestrel catches them is double increased when they are infected.

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